Comparison of the biologically effective UV in the shade for three action spectra

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Abstract
There have been numerous studies associated with the measurement of biologically effective UV (UVBE) irradiances for the solar zenith angles (SZA) observed during summer. However, only a small amount is known about UVBE levels related to the larger SZA seen during the autumn and winter months. Spectral UV irradiance measurements were made under clear sky conditions at a sub-tropical Southern Hemisphere site. Biologically effective UV levels for fish melanoma, DNA damage and cataract induction was investigated beneath four specific shade settings, for relatively cloud-free sky conditions and changing solar zenith angle (SZA) between 33^{o} to 63^{o}. The biologically damaging UV in the shade was significant for fish melanoma. Compared to that in full sun, the UVBE associated with DNA damage, cataract and fish melanoma were at levels of approximately 76, 78 and 65% respectively, beneath the shade umbrella. DNA damage, cataract and fish melanoma irradiance levels in the shade of a northern facing covered veranda were significantly less than those beneath the shade umbrella, with levels of approximately 19, 19 and 15% respectively. Although no action spectrum exists for human melanoma and cataract development, the fish melanoma and porcine cataract action spectra may provide an indication of the effective wavelengths. The relative UVBE in the shade compared to full sun depends on the action spectrum that is being considered. The reduction in the UVBE for the different action spectra is not related to the reduction in the unweighted total UV. Shade is important as a UV minimisation strategy, but for long periods, shade alone does not provide enough protection from some biologically damaging UV.

Keywords: Shade; UV; Melanoma; Cataracts; DNA damage.

INTRODUCTION

Australia has one of the highest rates of skin cancer incidence and mortality in the world, with two out of three Australians developing some form of skin cancer during their lifetime (ACCV, 1999; Roy and Gies, 2000). Melanoma is rated fourth of all cancers in Australia, as a cause of morbidity and mortality before the age of 70 (Green, 1984).

UV radiation is a carcinogen and repeated exposure to sunlight is now widely accepted as the major environmental cause of skin cancer and sun related eye disorders in all skin types who are genetically predisposed (Longstreth et al., 1995; NHMRC, 1996; Carter et al., 1999). UV-induced types of skin cancer include basal cell carcinoma, squamous cell carcinoma and melanoma. There is a clear relationship between UV dosage and the incidence of squamous cell skin cancer. Although sunlight exposure is implicated in melanoma development, the relationship with exposure is not completely certain as melanoma is not generally located on highly exposed areas of the body (Setlow et al., 1993; Preston and Stern, 1992; Urbach, 1997).

It is thought that intermittent severe exposures (severe enough to cause sunburn) are critical for UV-induced melanoma and that UV exposures in infancy are more dangerous than exposures later in life (Ambach and Blumthaler, 1993; Stanton et al., 2000). Although cutaneous melanoma is generally a disease of adulthood, children in Queensland, Australia, showed the highest incidence rates of melanoma in the world (Whiteman et al., 1997). Godar et al (2003) concluded from their work that individuals receive approximately 25% of their total lifetime UV radiation exposure by the age of 18.

Even though sunlight exposure is agreed as being one of the main etiologic agents in the development of malignant melanoma and cataracts in humans, the wavelengths responsible are not yet fully known (Setlow et al., 1993; Oriowo et al., 2001). If an estimate of the biological sensitivity of organisms to UV radiation is desired, responses to changes in wavelength dependence must be calculated (Jagger, 1967). Action spectra are used to show the relation between the irradiating photons and the effect on certain biological processes. Although no action spectrum exists for human melanoma, the fish melanoma action spectrum may provide an indication of the wavelengths effective in human melanoma development (Setlow et al., 1993; Gasparro et al., 1998). Research on cataract development using whole porcine lenses has also produced an action spectrum that may be used as an indicator of the wavelengths effective in cataract induction in the human eye (Oriowo et al., 2001).

The development of non-melanoma skin cancer as a result of induction by UVB radiation is accompanied by damage to the DNA and its repair system, and by an alteration of the immune system (Ambach and Blumthaler, 1993). DNA is believed to be the target because individuals defective in the repair of UV damage to DNA are more highly prone to the disease than the average person (Setlow et al., 1993). The UVB (280-320 nm) wavelengths of the DNA damage action spectrum are more effective than are the longer UVA (320-400 nm) wavelengths (Setlow, 1974). UV induced damage in DNA is repaired by a cascade of enzyme reactions and years of over exposure to UV cause errors to accumulate in replicated DNA (Urbach, 1997). These errors (mutations) can make certain genes, that are crucial for cell regulation, dysfunctional, and such an association was found in genes in human skin cancer (Urbach, 1997).

Shade is highly sought after to find solace from the sun's harmful rays during the day, but the levels of biologically effective UV radiation that exist in different shade environments are not fully understood. Past research has looked at the erythemal UV irradiances in various types of shade (Wong, 1994; Toomey et al., 1995; Moise and Aynsley, 1999; Parisi et al., 2000; Parisi et al., 2001; Turnbull and Parisi, 2003; Turnbull et al., 2003; Turnbull and Parisi, 2004a; Turnbull and Parisi, 2004b), but none have described a comparison of the biologically damaging UV associated with melanoma, DNA damage and cataract induction underneath specific shade structures. This research reports on the biologically effective UV that is associated with melanoma, DNA damage and cataract induction beneath four different shade environments during the months associated with autumn and winter for a Southern Hemisphere site. The months of March to August were selected because of the higher proportion of diffuse UV, caused by an increasing SZA, seen in the shade.

MATERIALS AND METHODS

Spectroradiometry

A scanning spectroradiometer fitted with a 15 cm diameter-integrating sphere (model OL IS-640, Optronics Laboratories, Orlando, FL, USA) that can be manually orientated was employed. For this research, the integrating sphere was 1.0 m above ground level. The spectroradiometer has a double holographic grating (1200 lines mm^{-1}) monochromator (model DH10, Jobin-Yvon, France) connected to a R212 photomultiplier tube (Hamamatsu Co., Japan) temperature stabilized by a Peltier cell temperature controller to 15.0 ± 0.5^oC. Stray light rejection is of the order of 10^{-8}.

Prior to each series of scans, the spectroradiometer was wavelength calibrated against UV mercury spectral lines and absolute irradiance calibrated against a quartz tungsten halogen lamp (250 W) operated at 9.500 ± 0.005 A d.c. and with a calibration traceable to the National Standards Laboratory at the CSIRO, Lindfield**.** The current was supplied to this secondary standard lamp from a regulated power supply (model PD36 20AD, Kenwood).

For each shade setting, measurements were at two specific periods of the day (11:30 - 12:30 Eastern Standard Time (EST), referred to as noon and 14:30 - 15:30 EST, referred to as afternoon) and for increasing solar zenith angle between 33^o and 63^o. The measurement protocol has been previously described (Turnbull and Parisi, 2003). Briefly, this involved: measurement of the UV spectrum in the sun on a horizontal plane (with the entrance aperture of the integrating sphere directed upwards) at a distance of 20 m or as far as possible from the shade; measurement of the UV spectrum in the approximate centre of the shadow cast by the shade structure; and then measurement of the UV spectrum in the sun a second time. The measurement planes were a vertical plane, horizontal plane and on a plane 45^o to the vertical for each of the shade settings. For the shade umbrella and sand pit, the measurements on the vertical and 45^o planes were directed towards the sun. For the covered veranda and covered walkway these measurements were aimed in a northern direction for two reasons: 1. the sun is to the north at solar noon; and 2. these positions gave the maximum irradiances in each case.

Day-to-day total column ozone levels were obtained from the TOMS (Total Ozone Mapping Spectrometer) web page. The data collected showed that the total ambient air ozone levels varied from 281 to 326 Dobson Units (DU) during the research period.

Shade structure description

The research was conducted using four different shade structures located at the University of Southern Queensland (USQ), Toowoomba (27.5^{o}S), Australia. The structures were a shade umbrella, a covered veranda, a covered sand pit and a covered walkway. These were selected because they are regularly used by the public and previous research (Turnbull and Parisi, 2003) found that there were relatively high erythemal irradiances under these shade structures. Brief details of the shade structures are described below:

- The diameter of the shade umbrella was 1.8 m and a height at the apex of 2.1 m. The ground cover was dry grass with an albedo of approximately 0.04;
- The northern facing veranda covering was approximately 7.0 m long, 1.5 m wide from the building wall and the eaves were 2.5 m high. A number of trees are located near this site and therefore have some influence over the scattered UV levels in the shade;
- The sand pit covering was 2.6 m in diameter, approximately 3.0 m high at the apex and 2.0 m high at the eaves. Trees, shrubs and a building are located near the shade structure. The albedo of the sand was approximately 0.1;
- The height of the walkway was approximately 4.0 m, the depth 2.5 m, length 6.0 m, and with an east/west path.

The shade umbrella and sand pit coverings were made from various woven materials. The transmittance through each of these were previously measured and reported (Turnbull and Parisi, 2003). Briefly, the UV transmittance of the shade umbrella material was determined with the scanning spectroradiometer on four separate occasions, scanning the incoming spectrum from 280 to 400 nm on a relatively cloud free day. The solar spectral UV irradiance, S(λ) was measured in 1 nm increments and then the shade umbrella fabric was placed directly over the opening of the integrating sphere in a stretched state similar to that when fully deployed. The spectral irradiance of UV passing through the fabric, $S_T(\lambda)$, was then measured and the UV transmittance, UV_T, was calculated using the following equation:

$$UV_T = \frac{\sum_{280}^{400} S_T(\lambda)}{\sum_{280}^{400} S(\lambda)} \qquad (1)$$

It was not practical to employ the scanning spectroradiometer to measure the transmittance of the covering material over the sand pit, because of the fixed nature of the shade structure being too high for the integrating sphere. As a result, the UV irradiances were measured in the shade with a radiometer (model 3D V2.0, Solar Light Co., Philadelphia, PA) fitted with a UVA detector and an erythemal UV (UVery) detector. The radiometer was calibrated to the spectroradiometer with the solar UV as the source. The transmittance measurements were made on relatively cloud free days by placing the entrance optics of the radiometer directly beneath the shade cloth (approximately 2.3 m above the ground), facing directly towards the sun. Full sun measurements were made by placing the radiometer at the same height, same orientation and approximately 1.5 m from the shade structure. The transmittance was calculated as the ratio of the shade measurements to full sun measurements. The UV transmittance through the material of the shade umbrella was calculated as being 0.5% for total UV and 0.9% for erythemal UV. There was no spectral dependency observed for the transmittance through the shade umbrella fabric. The erythemal UV through the material over the sand pit was equated to a transmittance of 4.8%, and with a corresponding UVA transmittance of 2.1%.

Biologically effective UV

To calculate the biologically effective UV irradiance in the shade, UVBE, the spectral irradiance, S(λ), may be weighted with the action spectrum for a particular biological process, A(λ), according to the following equation:

$$UVBE = \int_{UV} S(\lambda)A(\lambda)d\lambda \qquad (2)$$

For this research, the fish melanoma (Setlow et al., 1993), DNA damage (Setlow, 1974) and cataract (Oriowo et al., 2001) action spectra have been employed (refer to Figure 1). Linear interpolation was used for the fish melanoma and cataract action spectrums for wavelength points not present in the action spectra. Action spectra are normalized; therefore the plots provide a detailed change with wavelength for a specific effect, rather than an absolute quantity to compare between different action spectra.

RESULTS AND DISCUSSION

Biologically effective UV

Spectral irradiances taken beneath the four shade structures for a SZA of approximately 61^o have been weighted by the fish melanoma, DNA damage and cataract action spectra (Figure 1) and are shown in Figures 2a, b and c. Overall, the spectral irradiances generally increased with increasing wavelength from a cut-off wavelength between 295 and 305 nm, with maxima occurring at 400 nm. All spectral irradiance calculations are from 300 – 400 nm due to the high levels of noise in the shade irradiances below 300 nm. This may cause a slight underestimation in the UVBE levels for DNA damage and cataracts. The fish melanoma action spectrum (Figure 2a) illustrates that there is a significant biological response over the entire UV waveband, where as in Figures 2b and c the response is far more effective in the UVB waveband.

Table 1 shows the highest UVBE irradiance levels observed in the full sun and in the shade. The full sun and shade UVBE measurements are not relative to each other, they are only the maximums observed during the research. As can be seen from Table 1, UV irradiance levels associated with fish melanoma were significantly higher both in the full sun and in the shade compared to those for DNA damage and cataract induction.

UVBE levels for DNA damage, cataract and melanoma induction in the shade of the four structures for winter and autumn are plotted in Figures 3 and 4. All three measurement planes are graphed to show the variation of UVBE levels with the changing angle of the incident radiation. Overall, UVBE levels were the highest beneath the shade umbrella for all instances, where as the sand pit and covered walkway received the lowest irradiances especially in winter.

Shade ratios

Average shade ratios for DNA damage, cataract, fish melanoma and total UV, for relatively cloud free skies and two different times of day are shown in Table 2. The shade ratios are based on the angle (horizontal, 45^o and vertical) that received the highest UV irradiance in the shade. The shade umbrella received the highest relative proportion of

UVBE in the shade, as the SZA increased. Compared to that in full sun, the UVBE beneath the shade umbrella associated with DNA damage, cataract and fish melanoma were at levels of approximately 76, 75 and 65%, respectively for the larger SZAs.

The shade ratios in the shade for DNA damage and cataracts were greater than those for melanoma because of the higher relative effectiveness of the DNA and cataract action spectra in the UVB waveband compared to the UVA waveband. This results in a higher spectral effectiveness in the UVB compared to the UVA for the DNA and cataract action spectra. In comparison, the spectral effectiveness for the fish melanoma action spectrum is higher in the UVA compared to the UVB (Figure 2). As a result, due to the relatively higher degree of scattering of the shorter UVB wavelengths, the shade ratios for DNA damage and cataract induction in the shade are proportionally higher. Although the shade ratios for DNA damage and cataract induction are quite high in the shade, this does not necessarily translate across to meaning greater UV energy levels in the shade (refer to Table 1).

The northern facing covered veranda with surrounding trees received the lowest proportion of UVBE in the shade for an increasing SZA, with the highest levels at 22% for DNA damage, 19% for cataract and 15% for fish melanoma. Peak levels of UVBE beneath the sand pit covering were of the order of 44% for DNA damage, 33% for cataract and 26% for fish melanoma. UVBE levels beneath the covered walkway exhibited the sharpest increase for an increasing SZA, with maximum measured UVBE levels of 73% for cataract, 71% for DNA damage and 39% for fish melanoma. Once again, the shade ratios were higher for DNA damage than for melanoma, because of the increase in scattering of the shorter UVB wavelengths associated with the DNA damage action spectrum.

CONCLUSIONS

From this research it can be concluded that the relative UVBE in the shade compared to full sun depends on the action spectrum being considered. The reduction in the UVBE for the different action spectra is not related to the reduction in the unweighted total UV. DNA damage and cataract induction had the highest proportion of UVBE in the shade, but the lowest irradiances in the shade. To the authors' knowledge, this research is the first research to compare the UVBE for DNA damage, cataracts and fish melanoma in the shade.

When constructing shade structures, careful consideration must be used, because, even though summer has the highest UV levels in the full sun, winter has the highest relative proportion of scattered UV in the shade (Turnbull et al., 2003). Shade is important as a UV minimisation strategy, however for long periods, shade alone does not provide enough protection from some biologically damaging UV.

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Table 1. Select data of the highest UVBE levels observed in the full sun and in the shade for any shade structure. These are not corresponding full sun and shade measurements.

		UVBE (Wm^{-2})		
		Melanoma	DNA damage	Cataract
Full Sun	Autumn	15.01	0.0110	0.300
	Winter	12.84	0.0076	0.170
Shade	Autumn	5.59	0.0028	0.126
	Winter	5.87	0.0014	0.065

Table 2. Summary of average seasonal shade ratios with standard deviations in parenthesis for each shade structure and relatively cloud free skies.

		Seasonal Shade Ratios							
		DNA		Cataract		Melanoma		Total UV	
		Autumn	Winter	Autumn	Winter	Autumn	Winter	Autumn	Winter
Shade Umbrella	Noon	0.50(0.20)	0.49(0.26)	0.57(0.26)	0.45(0.16)	0.41(0.18)	0.33(0.08)	0.39(0.18)	0.31(0.07)
	Afternoon	0.70(0.10)	0.76(0.12)	0.75(0.13)	0.78(0.08)	0.51(0.05)	0.65(0.07)	0.49(0.05)	0.64(0.06)
Veranda	Noon	0.04(0.00)	0.22(0.01)	0.06(0.01)	0.13(0.04)	0.05(0.01)	0.10(0.04)	0.05(0.01)	0.10(0.04)
	Afternoon	0.14(0.03)	0.19(0.02)	0.14(0.04)	0.19(0.02)	0.11(0.04)	0.15(0.01)	0.11(0.04)	0.15(0.02)
Sand Pit	Noon	0.11(0.07)	0.44(0.14)	0.10(0.02)	0.33(0.05)	0.07(0.01)	0.20(0.04)	0.07(0.01)	0.19(0.04)
	Afternoon	0.27(0.03)	0.31(0.03)	0.23(0.02)	0.34(0.01)	0.14(0.02)	0.26(0.03)	0.13(0.01)	0.25(0.01)
Walkway	Noon	0.26(0.11)	0.35(0.06)	0.23(0.14)	0.41(0.18)	0.13(0.08)	0.26(0.03)	0.12(0.08)	0.25(0.07)
	Afternoon	0.71(0.13)	0.62(0.08)	0.69(0.09)	0.73(0.13)	0.39(0.04)	0.39(0.04)	0.37(0.09)	0.36(0.06)

Figure Captions

Figure 1. Action spectra for fish melanoma, DNA damage and cataract induction.

Figure 2. Spectral irradiances for the shade umbrella at a SZA of approximately 61^{o}, weighted by the fish melanoma (a), DNA damage (b) and cataract induction (c) action spectra for the shade umbrella (1), covered veranda (2), covered sand pit (3) and covered walkway (4).

Figure 3. Comparison of both autumn (black) and winter (white) UVBE levels on a clear day for the shade umbrella (a) and covered veranda (b), at both time periods and for the three orientations.

Figure 4. Comparison of both autumn (black) and winter (white) UVBE levels on a clear day for the sand pit (a) and covered walkway (b), at both time periods and for the three orientations.

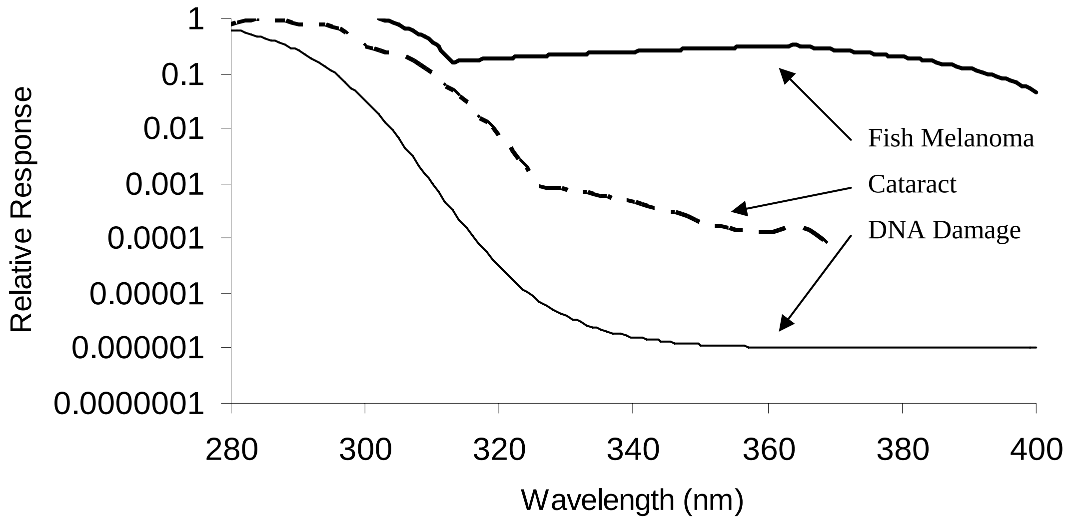

1
0.1
0.01
0.001
0.0001
0.00001
0.000001
0.0000001
Relative Response
280
300
320
340
360
380
400
Wavelength (nm)
Fish Melanoma
Cataract
DNA Damage

Figure 1.

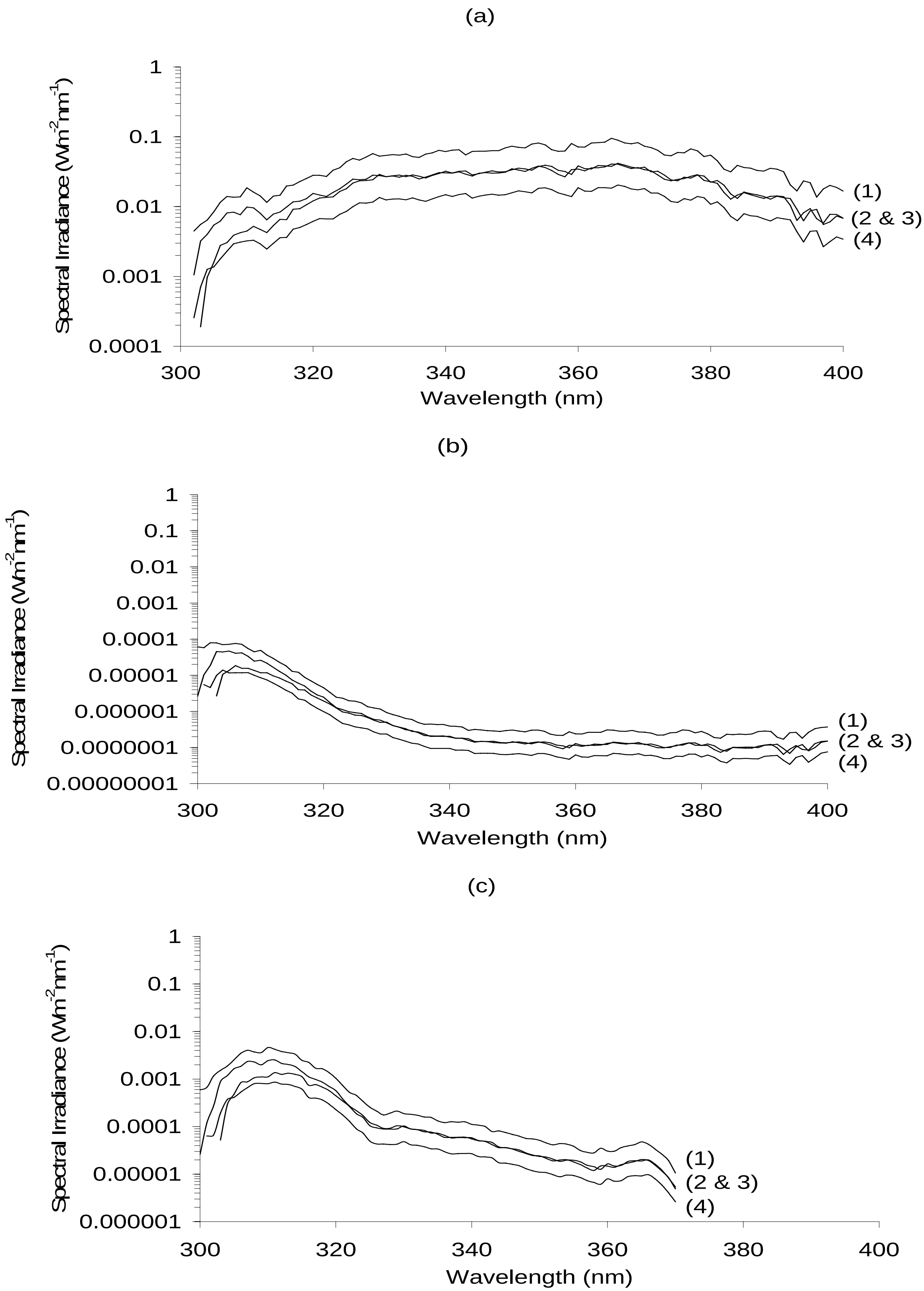
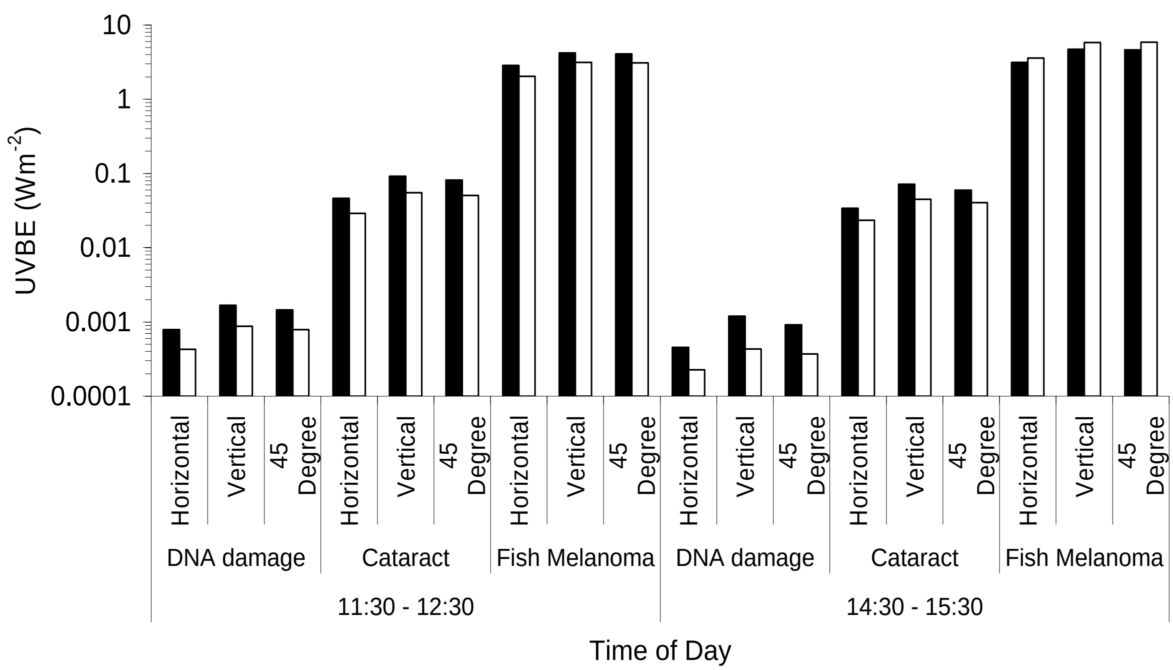

(a)
Spectral Irradiance (Wm-2nm-1)
1
0.1
0.01
0.001
0.0001
300
320
340
360
380
400
Wavelength (nm)
(1)
(2 & 3)
(4)
(b)
Spectral Irradiance (Wm-2nm-1)
1
0.1
0.01
0.001
0.0001
0.00001
0.000001
0.0000001
0.00000001
300
320
340
360
380
400
Wavelength (nm)
(1)
(2 & 3)
(4)
(c)
Spectral Irradiance (Wm-2nm-1)
1
0.1
0.01
0.001
0.0001
0.00001
0.000001
300
320
340
360
380
400
Wavelength (nm)
(1)
(2 & 3)
(4)

Figure 2.

(a)


10
1
0.1
0.01
0.001
0.0001
UVBE (Wm-2)
Horizontal
Vertical
45 Degree
DNA damage
Cataract
Fish Melanoma
11:30 - 12:30
14:30 - 15:30
Time of Day

(b)

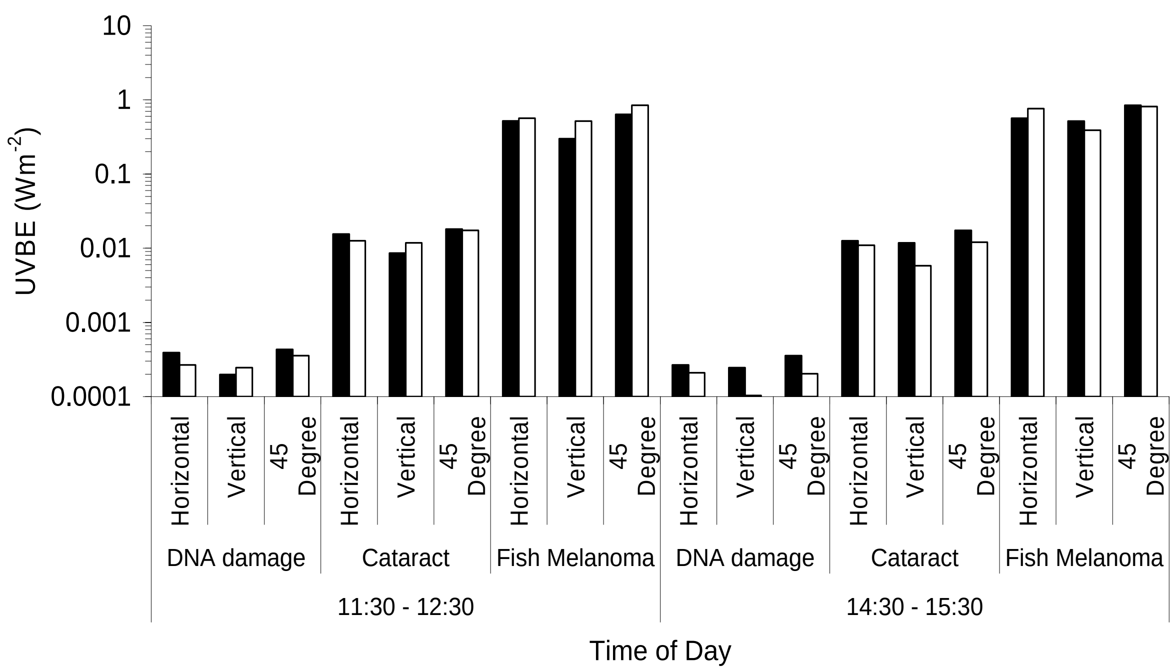
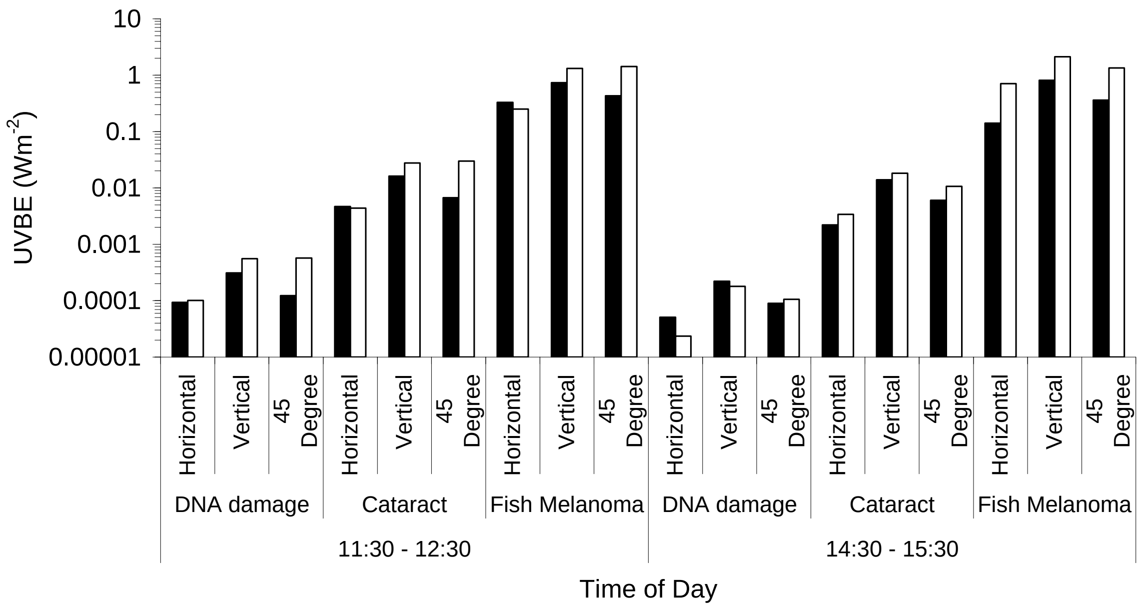

10
1
0.1
0.01
0.001
0.0001
UVBE (Wm-2)
Horizontal
Vertical
45 Degree
DNA damage
Cataract
Fish Melanoma
11:30 - 12:30
14:30 - 15:30
Time of Day

Figure 3.

(a)


10
1
0.1
0.01
0.001
0.0001
0.00001
UVBE (Wm-2)
Horizontal
Vertical
45 Degree
DNA damage
Cataract
Fish Melanoma
11:30 - 12:30
14:30 - 15:30
Time of Day

(b)

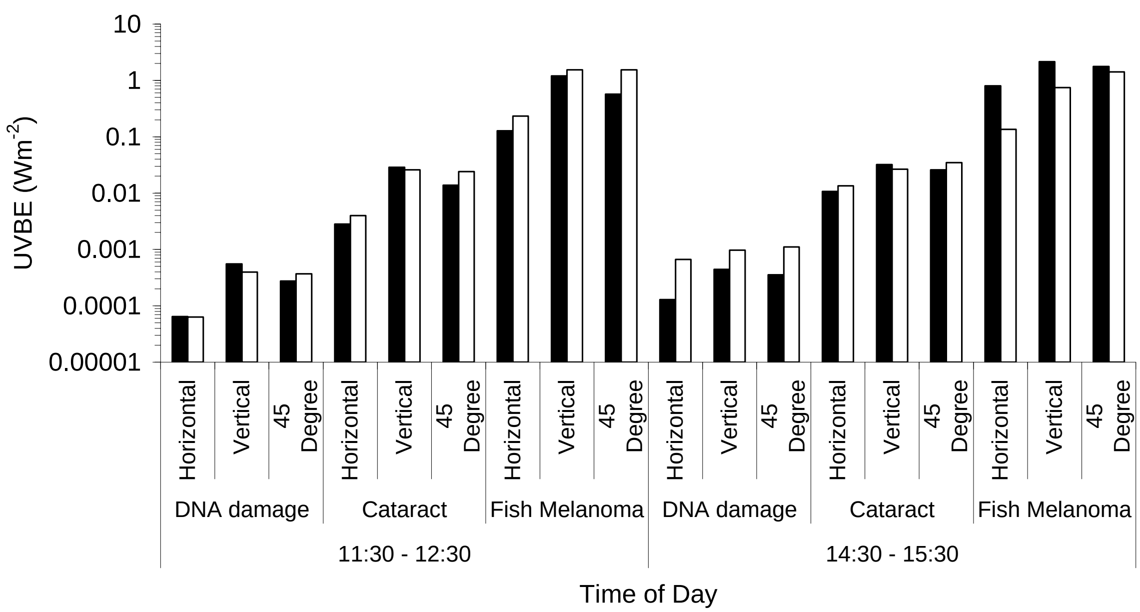

10
1
0.1
0.01
0.001
0.0001
0.00001
UVBE (Wm-2)
Horizontal
Vertical
45 Degree
DNA damage
Cataract
Fish Melanoma
11:30 - 12:30
14:30 - 15:30
Time of Day

Figure 4.